feels himself overcome with a species of lassitude and complains of being very tired. This state is very easily dissipated by either changing the mental attitude toward his task, or by permitting him to do something else. In some instances, however, lassitude will persist for a considerable time.

THERAPEUTIC IMPORT.

Although curious and interesting, relation of these cases would have little practical import for healing of the sick did differential diagnosis not provide an avenue to treatment of each case leading to improvement and recovery. Thus, the case of acute encephalitis was spared the risks of exploratory craniotomy, avoided an unnecessary saturation with antiluetics, and he was spared the expense of a rich diet.

The patient with post-encephalitic dystonia is saved from wasting his money upon useless manipulations whether they call themselves chiropractic or not, and is spared the annoyance and expense of being baked in a vain hope of relaxing his over-tight muscles.

The patient with a tumor of the frontal lobe is relieved by an operation upon the skull; he, too, is spared the infliction of mercury and arsenicals.

The woman with pellagra escapes the madhouse, useless medication, futile exploratory operation, and is quickly restored by appropriate diet.

The young girl with pituitary disease is spared the stigma of a perverse character, the trying discipline of psychomotor reëducation, and the murdering of the digestive organs by so-called tonics. She was soon restored to effective living by proper opotherapy.

The man who at first sight seems a wreck of his former self is spared the all too commonly imposed trip to Florida, a long stay in a sanatorium, or a life of permanent invalidism. He, too, is quickly restored to effectiveness by proper opotherapy.

The woman who has become a burden to her household, and whose illness is compelling her family to face an entire readjustment of their lives, is restored to her former effectiveness as director of her household and companion of her husband and children, but only after the nature of her trouble is ascertained to be due to a mere deficiency of thyroid secretions.

Of the torpid conditions ensuing upon psychic causes, of which an example would take too long to relate completely enough, the therapeutic import of a correct diagnosis is just as great; for it is especially to these patients that have been misdirected so often medicinal and physical measures without efficacy in such cases. Strychnine, forced feeding, rest, change of scene, douches, massage, treatment by cults—all fail to reach the source of the disturbance. The inertia of cases of this kind can be quickly and infallibly removed, by proper psychic means, and by them only. For if other means, in some few cases, seem efficacious, it is only because of the psychic accompaniments which some of them sometimes contain, and which may accidentally happen to fit the particular case; whereas intelligent psychotherapy ascertains and measures the situation and then meets it with a sure hand.

Most gratifying of all, the distress and ineffectiveness due to psychic disadaptations are permanently relieved by the rectification of the patient's mental attitude and accompanying emotion, with a success nearly invariable where proper means are used.

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## CONGENITAL MALFORMATION OF THE INTESTINE — ATRESIA AND IMPERFORATE ANUS.

### A REPORT OF TWENTY-SEVEN CASES.

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Congenital malformation of the intestine, although not by any means a common defect, is of sufficient importance to warrant the attention not only of the practising physician, but of the laity as well.

Recently while assisting Dr. Wolbach in autopsies at the Children's Hospital, Boston, two cases of malformation of the intestine were encountered in a single week. The similarity of sex, nature of defect and operation, and the fact that both died on the fourth day, led to my studies of this particular form of defect; with the result, I was able to collect twenty-five unpublished cases from the Warren Anatomical Museum of the Harvard Medical School.

In view of the fact that text books on diseases of children, obstetrics, and systems of surgery mention the condition from their respective points of interest, it is the object of the writer not merely to place these cases on record, but to include in this study such available information, other than those that are obvious at birth, that may be of interest on the subject.

I am indebted to Dr. Stone of the Surgical Service of the Children's Hospital for the clinical data on the first four cases; and to the Warren Anatomical Museum for allowing me to use the specimens and records; also to Dr. Wolbach for the assistance he gave me in the preparation of these cases.

REPORT OF CASES.

CASE 1.—A-21-19.

*Clinical Data:* Baby L., male, age 2 days, was admitted to the Children's Hospital, Boston, with imperforate anus. Although no meconium was passed by urethra, the symptoms were not

urgent, as there was no distention or vomiting. The family history was negative; there being no miscarriages, syphilis or tuberculosis.

A perineal exploration was done, but the rectum could not be located. On the following day a colostomy was done, after which the patient showed marked improvement, but of short duration. His temperature varied from 97 to 102 during his stay in the hospital. He died on the fourth day, two days after the operation.

*Pathological Report:* Necropsy was performed 9 hrs. post mortem by Dr. S. Burt Wolbach and Dr. W. S. Quinland.

*Anatomical Diagnosis:* Imperforate anus, persistent cloacal duct, congenital absence of left kidney, hypertrophy of the right ventricle of heart, hypertrophy of adrenal (left), colostomy wound, perineal wound, acute peritonitis.

*Body:* The body was that of a newly born male baby, measuring 42 cm. in length. At a distance of 2 cm. to the right of the umbilicus was a colostomy wound 5 cm. long. This wound was closed by sutures except in its middle portion, where there was an opening 1.5 cm. long, through which feces could be expressed. In the perineal region a gaping wound 2 cm. long and 4 cm. deep was seen to enter, the membranous urethra, instead of the rectum, which existed as a dilated sac high up in the pelvis.

*Peritoneal Cavity:* On opening the abdomen the stomach was seen to be distended with gas. The coecum and several loops of small intestine were deeply injected and matted together by fibrinous adhesions. The calibre of the gut was normal until the rectum was reached, where it became dilated to the extent of 7x5 cm., and communicated with the membranous urethra by an opening barely large enough to admit a small sized probe. The bladder was distended to its full capacity with dark brown turbid fluid, and its mucosa was deeply injected. Near the urethral orifice, two small openings were seen. One of these, situated somewhat to the right of the median line, communicated with the right ureter; the other, 1 cm. below and in the median line, proved to be a persistent cloacal duct. (Fig. 1.)

Other abnormalities present were a congenital absence of the left kidney and ureter. Hypertrophy of the left adrenal, which measured 5x4x.4 cm. A right kidney shaped like a mushroom with its ureter given off from the inferior (caudal) surface. The heart was flattened so that its transverse measurement was 4.5 cm. and its vertical 2.5 cm. A Meckel's diverticulum was present.

CASE 2.—A-21-20.

*Clinical Data:* D. M., male, age 3 days, entered the hospital with imperforate anus. He began to vomit greenish material at birth. His abdomen became distended, but there was no tenderness. His temperature varied from 97-101. The family history was negative, father, mother, and three other children being well.

A perineal exploration and colostomy was done. The rectum was not entered, and the patient died on the fourth day, 24 hours after the operation.

*Pathological Report:* Necropsy was performed 1½ hrs. post mortem by Dr. S. Burt Wolbach and Dr. W. S. Quinland.

*Anatomical Diagnosis:* Imperforate anus, patent cloacal duct, colostomy wound, perineal wound, acute peritonitis, acute cystitis. So nearly alike were the findings in this case to the preceding one, that a less detailed description is given.

*Body:* The body was that of a male infant 49 cm. long. The skin was lemon yellow. There was a colostomy wound 6.5 cm. long, and 4 cm. deep that terminated behind the neck of the bladder and 1 cm. below the dilated rectum.

*Peritoneal Cavity:* On opening the abdomen the intestine was seen to be deeply injected and covered with a viscous exudate. The stomach was distended. The lumen of the gut was normal, until the rectum was reached, where a dilatation of 6.5x3.5 cm. was encountered. This sac-like dilatation communicated with the prostatic urethra by a patent cloacal duct 2 mm. in diameter. (Fig. 2.) The mucosa of the bladder was markedly congested and oedematous. Other abnormalities were a bifid left thumb and much dense fibrous adhesions around the common bile duct.

CASE 3.—A-18-2.

*Clinical Data:* L. H., male, age 6 days, entered the hospital with persistent vomiting of dark foul material that started on the second day. The abdomen was markedly distended and tympanitic. The superficial veins of the abdomen were engorged, and active peristalsis could be seen through the abdominal wall. The external anus, which was patent, had many fissures around it. The family history was negative; father, mother and one other child were alive and well. An ileostomy was done, but the patient died very shortly after the operation.

*Pathological Report:* Necropsy was performed by Dr. S. Burt Wolbach.

*Body:* The body was that of a poorly nourished male infant measuring 54 cm. in length. At a distance of 2 cm. to the left of the umbilicus was a surgical wound 8 cm. long, with sutures intact.

*Peritoneal Cavity:* On opening the abdominal cavity, there was seen to be a complete break in the continuity of the gut so as to form a proximal and distal cul-de-sac in the middle third of the ileum. The proximal blind end was dilated to 3 cm. and had a surgical opening that was sutured to the abdominal wall. Connecting this proximal end to a collapsed similarly blind distal portion were two narrow glistening white cords of fibrous tisssue, each about 1 mm. thick and 3 cm. in length, and a portion of mesentery above. The distal part of the ileum was tightly contracted, but on incision was found to be

patent and empty. It led to a completely contracted coecum in approximately normal position. There was a normal appearing appendix coming from the posterior wall of the coecum. The ascending, transverse, descending colon, and sigmoid flexure and rectum were tightly contracted and empty, so as to resemble more nearly small intestine than large. This description of the defect is best illustrated in Fig. 3.

CASE 4.—A-16-4.

*Clinical Data:* S. B., female, age 3 days, was admitted to the hospital with abdominal distention and persistent vomiting of everything ingested since birth. No meconium was passed, although a patent anus which would not admit the tip of the fifth finger was present. Family history was negative. Father, mother and five children alive and well.

An enterostomy and perineal exploration was done. The rectum was, however, not entered and the patient died five hours after the operation. Here again, because of the close similarity of the defect in this case to that of the preceding, I shall only mention some of the more important findings.

*Pathological Report:* Necropsy was performed by Dr. Wolbach.

*Body:* The body was that of a female infant 43 cm. long, weighing 2400 grams. Above and below the anal orifice were surgical incisions 1 cm. long, and closed by sutures. An enterostomy wound 4 cm. long and 4 cm. to the left of the umbilicus was present.

*Peritoneal Cavity:* On opening the abdomen a defect quite similar to the preceding case was encountered in the upper third of the ileum. Following is a summary including the foregoing cases with twenty-three others collected from the Warren Anatomical Museum.

## DISCUSSION.

Although there are various developmental defects of the intestine and its related structures, namely, atresia, stenosis, imperforate anus, diverticula and cyst, I shall attempt only to discuss those conditions encountered in the cases of the present issue. For convenience I shall begin with imperforate anus; not merely because of the clearer and more general conception of its occurrence, but it is the condition most frequently met, and that which is less likely to escape the immediate detection of the accoucheur.

*Imperforate Anus:* The embryology in this instance is interesting. Quoting Gaston[6]: "About the first month, the invagination of the ectoderm of the skin takes place on the ventral side of the embryo, opposite to the terminal portion of the cloaca, by which the ectoderm is brought into contact with the entoderm of the lower end of the intestine, the two layers becoming the cloacal membrane. When the cloaca becomes sub-divided into the urogenital sinus and the rectum, the cloacal membrane is correspondingly separated into the urogenital aperture and into the anal membrane. About the end of the twelfth week the anal membrane ruptures forming the anus." Imperforate anus, then, results from imperfect union between the rectum above and the posterior part of the cloaca common to the urogenital aperture and the hind gut below. The deformity exists in varying degrees. The anal aperture may be separated from the rectum by mesodermal tissue an inch or more in depth as a result of insufficient invagination of the ectoderm to meet the rectum, which may end blindly above or communicate with the exterior by some unusual opening. Cases 6, 12, 15, 16, 19, 20 and 22 fall in this category. Another type is where the embryonal anal plate persists without the slightest invagination of the skin in this region. See Cases 1, 5, 10, 11, 13, 21, 23, 24, 25, 26, 29.

In the presence or absence of an external anus, if for some reason complete separation of the cloaca to form the urogenital sinus and rectum does not occur, the latter opens into the bladder, or more commonly into the prostatic or membranous urethra if the child be a male. See Cases 1, 2, 10, 13, 14, 26 and 27; or the rectum may open into the vagina if the child be a female. See Cases 11 and 25. From these types of imperforate anus, it can readily be seen how seven of these cases might have gone unrecognized at birth because of the presence of an external anus that did not communicate with the intestine above. Still more unusual openings of the rectum have been recorded in the literature; among them being two extraordinary cases cited by Bushe where in the case of Dinmore's an infant in whom the inferior portion of the abdomen was badly developed had the intestine directed upward, and opened under the border of the right scapula; and to that of Bils, in which the intestine mounted from the pelvis, through the chest, into the neck, and opened on the face by a very small opening.

## ATRESIA.

Although many hypotheses relative to the causation of atresia of the intestine have been propounded, among them being bands of adhesions, twists and bends of the gut, blocking of mesenteric vessels resulting in defective blood supply, etc., none of these seem to give a very satisfactory explanation for the complete break in the continuity of the gut in the small intestine. Primarily, the duodenum presents a well defined round lumen in embryos of about 7 mm. This lumen later becomes divided into compartments by septa of proliferating lining epithelium so that in embryos of about 14 mm. the lumen may be completely occluded. At 30 mm. the compartments begin to become confluent, and in this way a central lumen is re-established. The view most commonly held is that of Tandler[10] based upon the above briefly outlined embryology. Forssner[3] confirmed Tandler's views to the

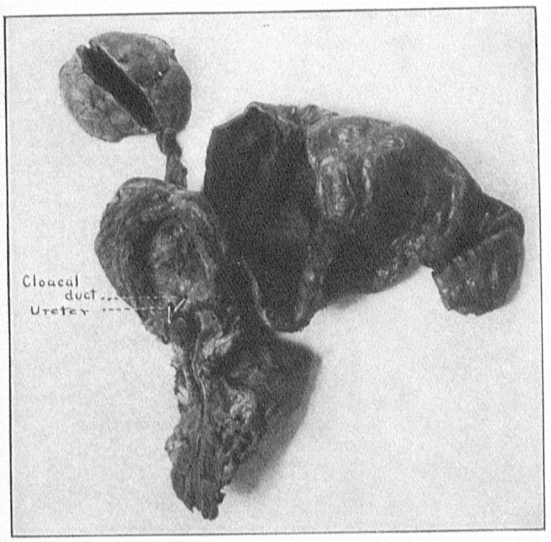


FIG. 1. Case 1. Ch. Hosp. A-21-19.—Imperforate anus, congenital absence of left kidney. Persistence of cloacal duct which enters bladder; on posterior wall median line, level of right ureteral orifice.

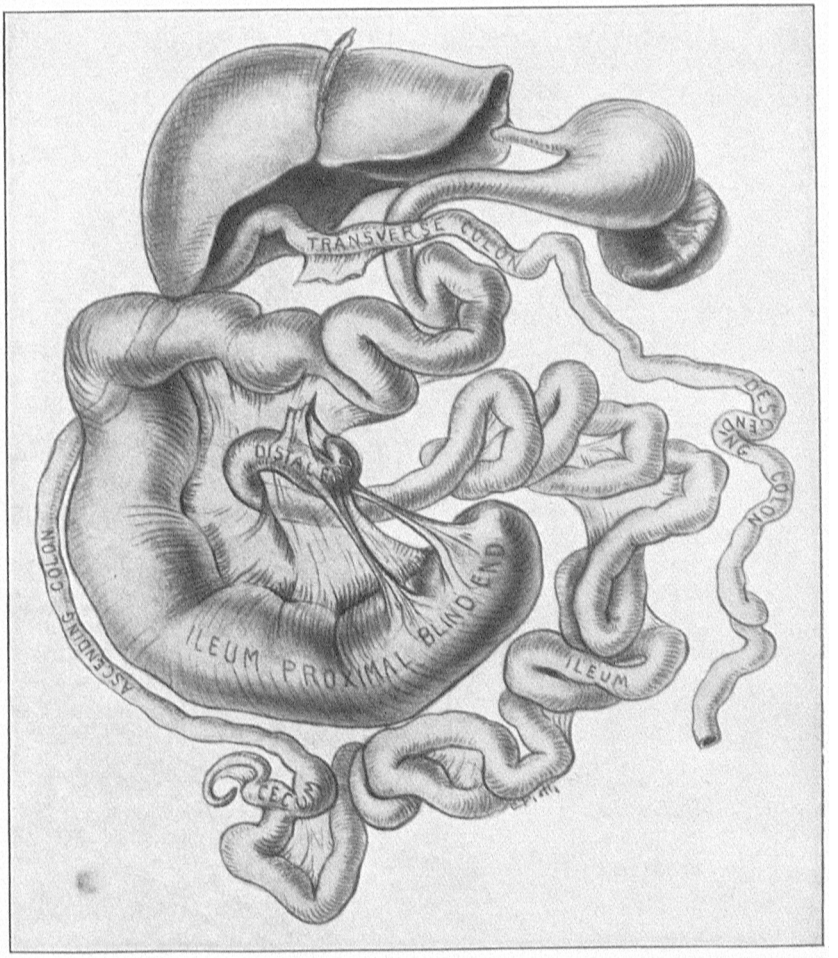


FIG. 3. Case 3. Ch. Hosp. A-18-2.—Congenital atresia in middle third of ileum, showing dilated proximal cul-de-sac and the contracted distal cul-de-sac attached to each other. The deformity falls in Group III (b) of Forssner's classification.

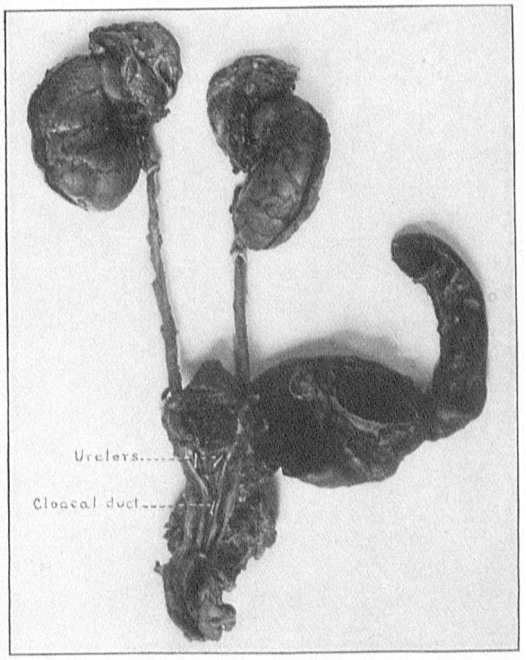


FIG. 2. Case 2. Ch. Hosp. A-21-20. Imperforate anus. Persistence of cloacal duct which enters prostatic urethra.

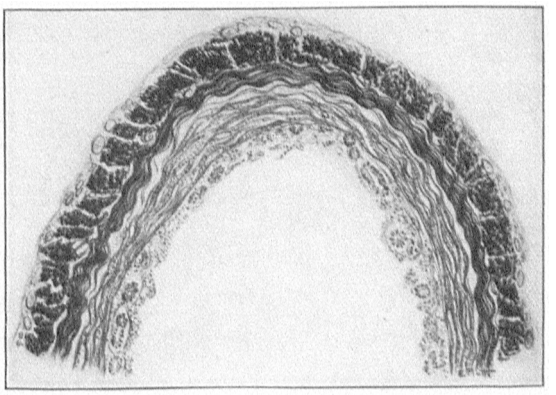

FIG. 4. Case 3. Ch. Hosp. A-18-2. Microscopic drawing of a longitudinal section taken through the distal cul-de-sac, showing no variation of the coats around the blind end.

| No. | Date | By whom presented | Sex | Malformation | | Treatment and Result | Remarks |
|---|---|---|---|---|---|---|---|
| | | | | Character-ization. | Anatomical Situation | | |
| 1 | 1921 | Dr. S. B. Wolbach Dr. W. S. Quinland | Male | Imperforate anus. | No external anus. Rectum ends as cul-de-sac 7x5 cm. having small opening that communicates with the prostatic urethra. Congenital absence of left kidney. Three lobes to left lung. | Operation - Perineal exploration. Following day a colostomy was done. Died on the fourth day, two days after the operation. | Meckel's diverticulum was present. Patient showed marked improvement of short duration immediately after the colostomy. Autopsy showed acute peritonitis. |
| 2 | 1921 | Dr. S. B. Wolbach Dr. W. S. Quinland | Male | Imperforate anus. | No external anus. Rectum ends in cul-de-sac 6.5 x 3.5 cm. having small opening in prostatic urethra. | Operation - Perineal exploration and colostomy. Died on the 4th day, 24 hours after the operation. | Patient drained freely during the night following operation, but rapidly grew weaker. Stimulants were used without success. Autopsy showed acute peritonitis. Rectum not entered by perineal route. Bifid right thumb. |
| 3 | 1918 | Dr. S. B. Wolbach | Male | Atresia | Ileum ends in cul-de-sac in middle third. Remainder of intestine is tightly contracted. | Operation - Ileostomy. Died on 6th day shortly after the operation. | There were several small fissures about the anus. |
| 4 | 1916 | Dr. S. B. Wolbach | Female | Atresia | Ileum ends in cul-de-sac just below jejunum where it is 6 times the width of the large intestine which measures .7 cm. across. Anus is patient. | Operation - Enterostomy. Perineal exploration. Died 5 hours after operation. | Anus although patent, will not admit the tip of the little finger. Probe can be inserted through anus for only 1½ cm. The rectum was not entered at operation. |
| 5 | | | | Imperforate anus. | Rectum ends blindly above anus. No external anus. | | Museum specimen - No record. |
| 6 | | Dr. S. M. Stone | Male | Atresia | Rectum ends in cul-de-sac 3 cm. above the anus which is of normal size. | Operation. Trochar was passed on the eighteenth day. Died on the 24th day. | Rectum and anus were separated by a small amount of tissue. No opening between bladder and urethra. |
| 7 | 1895 | | | Atresia | Duodenum ends in cul-de-sac 5 cm. wide. Shreds of mesentery connect duodenum with intestine below which measures only 5mm. wide. Constriction below pylorus. Esophagus dilated. | Died on sixth day. | |
| 8 | 1892 | Dr. Garnett | | Atresia | Duodenum ends in cul-de-sac and is enormously dilated 6.5 cm. Rest of intestine well formed and collapsed. Blind end of jejunum .7 cm. wide. | Died on third day. | Child at term. |
| 9 | 1889 | Dr. H. E. Marion | | Atresia | Duodenum ends in cul-de-sac and dilated 4 cm. Bile duct empties just below into otherwise well formed intestine. | Died on seventh day. | |
| 10 | 1877 | Dr. S. M. Stone | Male | Imperforate anus. | No external anus. Rectum opens into bladder. Ureters dilated and course along the outside of bladder to the penile urethra. Penis rudimentary, scrotum divided. | Died. | There was a structure corresponding with the seat of the verumontanum. |
| 11 | 1877 | | Female | Imperforate anus. | No external anus. Rectum ends blindly with small opening into vagina. | Operation - Died. | |
| 12 | 1877 | | Male | Atresia | Rectum ends blindly above the anal orifice from which it is separated by fibrous tissue. | Operation - Died. | |
| 13 | 1877 | | Male | Imperforate anus. | No external anus. Atresia of rectum with opening in prostatic urethra. | Operation. Perineal exploration. Died. | |
| 14 | 1877 | | Male | Imperforate anus. | No external anus. Rectum dilated and enters the membranous portion of urethra. | Operation. Perineal exploration. Died. | |
| 15 | 1869 | | | Imperforate anus. | Rectum ends in cul-de-sac a few cm. above anus. | Operation. On the 4th day a trochar was passed. Opening was enlarged. Died on the 6th day. | Meconium passed freely after the use of trochar, which passed through the surrounding tissue into peritoneal cavity. |
| 16 | 1858 | Dr. James Ayer | Female | Atresia | Rectum ends in cul-de-sac separated by narrow bands of tissue from anus which is formed. | No operation. Died on sixth day. | Second child in the family with atresia of the intestine. The previous one, a male, lived 8 days, during that time he passed meconium per urethra. |
| 17 | 1855 | Dr. Seaverns | | Atresia | Anus. At birth a small probe only could be passed. But it became somewhat dilated later. Intestine was dilated above. | No operation. Died at 18 months | |
| 18 | | | | Atresia | Duodenum, ends as dilated cul-de-sac 4.5 cm. wide. Not connected with ribbon like intestine of .5 cm. width. | No operation. Died on 5th day. | |
| 19 | 1848 | Dr. b. Porterman | Male | Atresia | Patent external anus that admitted the finger to a depth of half an inch. Rectum about 3 cm. above. | Operation. Anus was further opened by incision 1½ inches upward. Died few hours later | The intestine was not entered at operation. |
| 20 | 1847 | Dr. A.B. Moore | Female | Imperforate anus. | Atresia of Rectum, two above anus. Separated from 1 inch of rectum immediately above anus by less than an inch. | Operation. Trochar was passed through the anal plate for about 2 inches on the third day. Died two days later. | The trochar failed to enter the intestine. |
| 21 | 1847 | Dr. J. C. Warren | | Imperforate anus. | Atresia of ascending colon. Coecum measures 5 x 3 cm. Attached distally is a blind atrophic end 3 x .6 cm, representing the remainder of the large intestine. | Died in a few days. | |
| 22 | 1846 | Dr. John Flint | Male | Atresia | Patent external anus with one half inch of intestine above. Upper intestine ran a straight course and terminated in cul-de-sac about 4 cm. above anus. | No operation. Died on the sixth day. | Operation was not done possibly because there was a patent external anus. |
| 23 | 1836 | | Female | Imperforate anus. | No external anus. Atresia of rectum above the anus. | Perineal exploration. Died 17 hours after. | The posterior parieties of the vagina were extensively wounded, resulting in profuse hemorrhage. Intestine not entered. |
| 24 | 1840 | | Male | Imperforate anus. | No external anus. Rectum ends in cul-de-sac a short distance above the anus. | No operation. Died on the fourteenth day. | The lower portion of the small intestine and the whole of the large were inflamed. Large intestine was ulcerated. |
| 25 | 1839 | Dr. Wolker | Female | Imperforate anus. | External anus absent. Rectum ends in cul-de-sac about 4 cm. above anus, and communicates with upper posterior vagina by small opening. | Colostomy. Died at 3 months. | Two or three weeks before death the colostomy wound healed and feces was passed as before by the vagina. |
| 26 | 1837 | Dr. J. H. Lane | Male | Imperforate anus. | No external anus. Rectum dilated and ends in cul-de-sac with capillary opening into the membranous urethra. | No operation. The patient was kept under the influence of paregoric. Died on the ninth day. | Developed broncho-pneumonia as a terminal infection. |
| 27 | 1810 | Dr. W. G. Chamberlain | Male | Imperforate anus. | No external anus. Rectum ends in cul-de-sac with minute opening into the membranous urethra. Penis rudimentary. No scrotum. | Died. | No available record of treatment. |

effect that a condition of more or less complete occlusion of the duodenum may persist in the human from the very early embryological stage (5 mm. to 30 mm. in embryos), in which it normally occurs, until birth. The persistence of the epithelial septa is followed by an ingrowth of mesenchyma. Forssner found such atresias in the human colon and esophagus. He confirmed Tandler's work. Kreuter[3] also finds it in the colon. Forssner[4] quotes numbers of authorities giving such epithelial occlusions of the intestinal tract in various animals,—fowls, frogs, guinea-pigs, rabbits, dogs, and swine. He confirms these findings in the normal embryo of man's duodenum, rats' and fowls' duodenum, as the above sequence in the development of the intestine. He gives the following classification of the defect.

Group I. Septa of mucous membrane (a) with epithelium wholly intact. (b) Loss of epithelium in places (irisform).

Group II. Blind ends connected with a strand composed of serosa, muscularis and submucosa. In the submucosa one finds (a) No epithelium. (b) Only an occasional epithelial rest. (c) A small epithelial tube joining the blind ends.

Group III. Free blind ends (a) Not attached to each other. (b) Attached to each other.

The etiological factor of intestinal atresia and stenosis is to be found in the embryonal occlusions. One can interpret the subsequent pathogenic development as follows. If the epithelial occlusion does not disappear when the fold-building process through the ingrowth of the mesenchyma begins, then the ingrowth of the mesenchyma into the epithelium occurs before the continuity of the tube is re-established. (A partition.) If the connective tissue bridge is thin, then it provides a membrane. If it is thicker, it becomes a cord. If the connective tissue bridge is not complete or is very delicate at one place so that it tears, an incomplete membrane is formed. Through modification occurring during fetal life the various forms of atresia and stenosis can occur. It is possible to have a malformation which originally was a stenosis develop into atresia, which accounts for the fact that meconium is sometimes found distal to the atresia, if such a defect is formed after the third month. Atresia of the esophagus is to be explained in the same manner, but in addition the very early communication between the esophagus and trachea must be taken into account. It is further stated, that the gut fails to expand owing to the large size and downward growth of the liver at this stage, and to pressure exerted by the mesodermal wall on the closed epithelial cord.

In order to determine as nearly as possible in my cases what histological changes had occurred in places where the gut was occluded, I made longitudinal and cross sections through the terminal portions of the proximal and distal cul-de-sacs of Kaiserling fixed preparations. The sections were stained with hematoxylin and eosin. Microscopically none of the sections showed any evidence of scarring in the coats, no intervention of fibrous tissue or proliferation of epithelium between the walls of the gut or anything that would suggest a healed inflammatory process. On the contrary, the mucosa, submucosa, muscle and serous coats were seen in longitudinal sections to course without any apparent variation in thickness from one wall of the intestine around the blind end, to the opposite wall. These observations, which are quite consistent with those of Forssner,[5] were best studied in sections taken from the distal cul-de-sacs where there was absence of dilatation. (See Fig. 4.) Sections taken from the proximal cul-de-sacs showed the above changes with less distinctness, due on the one hand to stretching of the coats, and on the other to a localized acute inflammation either as a result of accumulated meconium, or an enterostomy operation. In some of the cases the walls of this portion of the gut were definitely thickened and discolored dark red. Microscopically, sections from the thickened wall showed hypertrophy of the muscle coat and irregular losses of mucosa. Sections of bands of fibrous tissue (apparently mesentery) that joined the proximal and distal cul-de-sacs were also examined microscopically to determine if possible to what extent thrombosis plays in the formation of such defects; but no blood vessels could be found in any of the sections. From these observations and the works of other investigators, I am inclined to believe that the break in the continuity of the gut is due to embryonic arrest in development, rather than to some mechanical injury of which it may be the end-result.

## OCCURRENCE.

*Location:* Atresia of the intestine may occur in any portion of the intestinal tract. In twenty of the cases herein presented it occurred in the rectum; once in the ascending colon, twice in the ileum (first and middle thirds), and four times in the duodenum. From so few cases as these, it is wholly impossible to arrive at any definite conclusion as to what portion of the intestine the defect is most likely to occur; yet, statistics record the majority of cases to occur in the duodenum and the jejuno-ileum regions. Just how many of these cases occur each year it is difficult to say with any degree of certainty, since it may be assumed that a few are likely to go unrecognized; but judging from the figures given below it may be concluded that the condition is one of the rare forms of congenital malformations. I am indebted to Drs. F. S. Newell, Boston Lying-In Hospital; J. B. DeLee, Chicago Lying-In Hos-

pital and Ross McPherson, New York Lying-In Hospital, for the following data. In the last 10,000 confinements at the Boston Lying-In Hospital, only one case of imperforate anus was encountered.

In 10,000 cases at the Chicago Lying-In Hospital two cases were encountered. In 50,000 cases at the New York Lying-In Hospital, there were 28 cases of atresia and imperforate anus; 24 of these were males and 4 were females.

### PROGNOSIS.

The prognosis is usually grave on account of complications; hemorrhage, peritonitis and septicemia. According to Bevan,[2] because of the probability of an ascending nephritis developing, it is usually wise in these cases of imperforate anus where there is connection between the lower end of the rectum and the bladder, to be satisfied with a permanent colostomy opening, and not risk the life of the patient in an attempt to separate the rectum and the bladder. If, however, the child be a female, the prognosis is more favorable, particularly if the recto-vaginal opening is of fairly large size. Gay cites a case of Morgagni in which Tatonicus, a Jewess, passed all her feces by the vagina and lived to the age of 100 years. Of Dr. Ross McPherson's 28 cases, 17 died, 10 lived, and one was a stillbirth. Death occurs usually in 5 or 6 days if the occlusion is high up in the small intestine. Atresia and imperforate anus are sometimes complicated by other anomalies that may or may not influence the prognosis of the case. This depends largely on the location and extent of such a defect. See Nos. 5, 2, 10 and 27.

### TREATMENT.

Surgical invention is the only treatment. Because of the variation in defect, the procedure must necessarily be modified to best meet the requirements of the existing condition. The perineal approach, which is the one frequently adopted, is sometimes futile on account of the very limited incision through which the surgeon is compelled to explore the deeper perineal structures, in an attempt to find the rectum, which might terminate high up in the pelvis. In such an event, an enterostomy is resorted to.

The antiquity of the operation as given by Thom[9] dates from eighteen hundred years ago; the first recorded instance being that of Egineta. Bell of England, in 1787, was the first to adopt the rational procedure of dissecting through the perineum and searching for the rectal ampulla. The first successful operation by this method in the United States was performed by Campbell, 1790. While to Amusat of Paris in 1835, and somewhat later to Verneuil, belongs the honor of perfecting the operation as it is now performed; *i.e.*, dissecting through the perineum without injury to the sphincter muscles, searching for the rectum, opening it, and suturing the bowel to the perineal incision.

### SUMMARY.

Atresia and imperforate anus may be regarded as rare forms of congenital malformation of the intestine due to embryonic arrest in development. The defect occurs once in about 15,000 infants, and may be found in any portion of the intestine from the duodenum to the rectum.

It is uncertain whether the malformation is in any way influenced by hereditary tendencies, since it is impossible to arrive at definite conclusions from so few cases as these. Only one of this collection, No. 16, gave a history of another child in the family having passed feces per urethra. The prognosis is grave on account of complications, particularly in males, in which sex the malformation is more frequently present. Of these 27 cases 13 were males, 6 were females, and in the remaining 8 the sex was not given. The only treatment is surgical.

Ordinarily, the presence of a patent external anus furnishes enough proof that the intestine is well formed; but from the foregoing observations it is obvious that one cannot emphasize too strongly the importance that should be attached to the first bowel movement in the new-born, since a break in the continuity of the gut is sometimes present.

It is of further interest to note that in the following cases no operation was performed.

No. 16—Atresia, died on the 6th day.
No. 18—Atresia, died on the 5th day.
No. 22—Atresia, died on the 6th day.
No. 24—Imperforate Anus, died on the 14th day.
No. 26—Imperforate Anus, died on the 9th day.

The fact that these five cases lived over a period of from five to fourteen days without operation, makes it more apparent that early operation in every instance should be resorted to as a means to a more favorable prognosis.

#### REFERENCES.

1. Bushe, Geo. Diseases of Rectum, etc., 1837, 46.
2. Bevan, A. D. S. Clinics 1920, IV, 80.
3. Forssner. Anatomische Hefte 1907, XXIV, 7.
4. Forssner. Ibid., page 8.
5. Forssner. Ibid., page 105.
6. Gaston, J. M. International Clinics 1919, III, 217.
7. Gay, Geo. H. Boston Med. and Surg. Jour., 1871, LVII.
8. Kreuter. Habilitationsschrift Erlangen 1905.
9. Thom, B. P. Medical Record 119, XCI, 678.
10. Tandler. Morphol. Jahrb., XXIX, 187.

## TREATMENT OF DIABETIC GANGRENE.

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GANGRENE occurring in the elderly diabetic patient has long been regarded with discouragement by both surgeon and physician. The tedious, if not eventually unsatisfactory, course